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Prevalence and predictors of externalising behavior in young adult survivors of pediatric traumatic brain injury.

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Abstract

Objectives: To investigate rates of clinically significant externalizing behavior in young adult survivors of pediatric TBI, and evaluate the contribution of pre- and post-injury risk and resilience factors to externalizing behaviour outcomes 16 years after injury.

Setting: Melbourne, Australia

Participants: Fifty-five young adults (*M* age = 23.85; Injury Age: 1.0 - 12 years) admitted to an emergency department following TBI between 1993 and 1997.

Design: Longitudinal prospective study with data collected at the acute, 10-year and 16-year post-injury time points.

Main Measures: Severity of TBI, adaptive functioning, family functioning, full scale IQ, executive functioning, social communication, and symptoms of externalizing behavior (EB).

Results: One of every four young people with a history of pediatric TBI demonstrated clinical or sub-threshold levels of EB in young adulthood. More frequent externalizing behavior was associated with poorer pre-injury adaptive functioning, reduced full scale IQ and more frequent pragmatic communication difficulty.

Conclusion: Pediatric TBI is associated with elevated risk for externalizing disorders in the transition to adulthood. Results underscore the need for screening and assessment of TBI among young offenders, and suggest that early and long-term targeted interventions may be required to address risk factors for EB in children and young people with TBI.

Keywords:

Traumatic brain injury, externalising behavior (EB), crime, antisocial behavior, young adulthood

Introduction

A growing number of research studies, undertaken within various national contexts, consistently demonstrate a disproportionately high prevalence of traumatic brain injury (TBI) amongst youth and adult offending populations. Studies estimate that between 12% and 24% of the general population have experienced a head injury resulting in loss of consciousness[1-4]. This compares to equivalent reported rates of between 32% and 46% amongst young people in custody [5-8], with several studies suggesting over 60% of adult prisoners have experienced a head injury with loss of consciousness [9-11].

Studies of adult offending populations seldom indicate whether TBI was sustained in childhood or adulthood. Nonetheless research has repeatedly demonstrated that 'life-course persistent' offending typically begins in childhood and is commonly associated with 'neurocognitive impairments' [12,13]. This would suggest that children experiencing pediatric TBI are at elevated risk for persistent offending beyond adolescence, and that their experiences run counter to the norm of desistence from criminality during young adulthood [14,15]. Despite this apparent association between TBI and persistent adult offending, to date there is insufficient research regarding such behavior in the transition into young adulthood amongst those who experience pediatric TBI.

There is considerable evidence that pediatric TBI contributes to impairments in executive functions, cognitive skills and EB (such as aggression, hyperactivity, bullying and defiance) that are commonly identified as risk factors for antisocial behavior and criminality. TBI commonly involves pathology to anterior brain regions implicated in executive functioning, as well as traumatic axonal injury that may contribute to deficits in social cognition, attention, learning difficulties and pragmatic communication [16-18]. Impairments in executive functioning have been detected soon after injury and appear to persist or even worsen with time since injury [19-21], likely reflecting a failure to develop and acquire skills at

an age appropriate rate [22]. It may be that such deficits contribute to antisocial or offending behavior through decreased inhibition, poor anticipation of the consequences of specific actions, or an inability to recognise when certain behavior is inappropriate in a given social context [23,24].

Reduced cognitive empathy implies an inability to see the consequences of antisocial behavior or to empathize with victims [25]. These deficits are a common consequence of pediatric TBI, particularly among children with frontal injuries [26]. In addition, children and adolescents with TBI experience persisting difficulty interpreting non-verbal emotion cues from facial expressions and prosody, as well as impairments in social or pragmatic communication [22,27]. These deficits are likely to contribute to reduced interpersonal effectiveness, which may in turn lead to frustration and distress, reflected in EBs that are shown to persist or even worsen with time since injury [28-31].

Though persisting injury-related neurocognitive impairments may elevate risk for antisocial or offending behavior after TBI, criminological research suggests that such injuries may influence offending via exposure to social and environmental experiences that may exacerbate the neurological consequences of brain injury[32-34]. For example, studies have linked permissive or authoritarian parenting styles, poor parental mental health and lower socio-economic status to long-term behavioral problems following TBI [35-38]. Persistent problems in academic performance, including specific difficulties in reading, spelling and arithmetic are commonly reported after TBI [39-42], and are likely to have a cumulative impact on educational opportunities, leading to challenges in engaging in later stages of education, particularly in the transition to secondary school [43-46].

The Heuristic Model of Social Competence (HMSC) [47] provides a useful framework for conceptualizing how such a range of injury and non-injury related risk and resilience factors may contribute to variability in social functioning after TBI, and can therefore be

usefully applied to the study of crime and antisocial behavior. Injury related factors, including injury severity and pathology location, are conceptualized as factors that increase the likelihood of deficits in social cognition and atypical social interaction, while environmental factors may further heighten risk or represent sources of resilience that buffer against the negative long term consequences of TBI. In addition, the model posits reciprocal interactions between various components of social information processing (SIP; cognitive-executive functions, social cognition, social problem solving), social interaction, and social adjustment, such that deficits in any one component in SIP may contribute to impaired social interaction and poor social adjustment, including EB.

In order to address limited understanding of the links between criminality and pediatric TBI, further research is required to investigate factors that may contribute to maladaptive or antisocial behavior among young adult survivors of pediatric TBI. In conceptualizing EB as a marker of risk for delinquency and/or criminal behaviour, it may be that injury-related neurocognitive impairments and environmental factors confer risk for criminality via their influence on EB.

The objectives of the present investigation were to (1) examine the prevalence of clinically significant EB problems in young adult survivors of pediatric TBI and (2) evaluate the respective contributions of a variety of injury and non-injury related risk and resilience factors at various time points to variability in EB outcomes 16 years after pediatric TBI. More specifically, guided by the HMSC model[47], we aimed to examine relationships between long-term externalizing symptoms and a variety of individual and environmental factors including: pre-injury individual and family functioning; injury severity and acute intellectual functioning; executive functioning; and social cognitive and affective functions, including social perception and pragmatic communication.

We predicted that relative to population expectations, a significantly greater proportion of young adults with TBI would show clinically significant EB. Furthermore, we hypothesized that more frequent EBs would be related to (a) greater injury severity; (b) poorer pre-injury adaptive and family functioning; (c) poorer acute intellectual functioning; (d) poorer executive function at 10-years post injury; and (e) reduced emotional perception and more frequent pragmatic communication difficulty at 16-years post-TBI.

Method

Participants

This longitudinal study followed up a sample originally recruited from consecutive admissions to the emergency department at The Royal Children's Hospital, Melbourne, Australia (RCH), between 1993 and 1997 for traumatic brain injury [48]. Inclusion criteria for the original study were: (1) age at injury 1.0 to 12.0 years; (2) documented evidence of TBI, including a period of altered consciousness; (3) sufficiently detailed medical records for diagnosis of injury severity. Exclusion criteria were: penetrating or non-accidental head injury; history of previous closed head injury; or pre-existing physical, neurological, psychiatric or developmental disorder.

During the initial recruitment period, 172 children aged under 12 years were admitted to hospital with a diagnosis of TBI and participated in the initial data collection. At 16 years post-TBI, 66 participants could not be located, and 38 declined to participate (not interested / too busy), 2 were deceased and 11 had incomplete datasets. Thus for the current paper 55 young adults (m age = 23.82 years; range = 16.25 - 30.58 years; SD = 3.85) from the original TBI sample yielded data across the acute, 10-year and 16-year time points and were included in analyses. Participating and non-participating samples were compared on demographic and injury characteristics, including socio-economic status, gender, age at injury and the

length of period of altered consciousness, to examine potential biases in the 16-year follow-up sample, with no significant differences identified (p>.05).

Measures

Details of the child's medical and developmental history, and family demographic information were collected at study enrolment. Severity groups were derived from a combination of measures, including period of altered consciousness on the Glasgow Coma Scale (GCS) [49], and presence of radiological and neurological abnormalities. This resulted in the following groups: (i) mild TBI (n = 15): GCS on admission 13–15, no evidence of mass lesion on CT/MRI scans, and no neurologic deficits; (ii) moderate TBI (n = 29): GCS on admission 9–12, and/or mass lesion or other evidence of specific injury on CT/MRI, and/or neurological impairment; and (iii) severe TBI (n = 11): GCS on admission 3–8, and mass lesion or other evidence of specific injury on CT/MRI, and/or neurological impairment.

Pre-injury.

The *Vineland Adaptive Behavior Scale* (VABS) [50] provides parent report of a child's level of adaptive function and was collected at time one to represent the child's pre-injury functioning. The present study utilised the Total Adaptive Behavior score (M = 100, SD = 15), and the Daily Living Skills and Socialization indexes as measures of pre-injury function.

Pre-injury family environment was measured using the parent-report *Family Functioning Questionnaire* (FFQ) [51]. Each item was rated on a 6-point scale where 1 = totally agree to 6 = totally disagree. Three factors are derived from the measure: Conflict, Intimacy and Parenting Style, with higher scores reflecting more of that characteristic. The Intimacy factor was utilised for statistical analyses because it represents a measure of family cohesion [52].

174

175 *Acute post-injury.*

176 The IQ assessment employed at the acute time point varied depending on the child's
177 age. Thus *Bayley Scales of Infant Development* children aged <30 months; *Wechsler*
178 *Preschool and Primary Scale of Intelligence Revised* [54] for children aged 30 months to 6.5
179 years; and *Wechsler Intelligence Scale for Children - Third Edition* [55] children aged >6.5
180 years were all used. Full scale IQ scores (FSIQ), were used in analyses (M = 100, SD = 15).
181 *Daniel's Scale of Occupational Prestige* [56] was used as a rating of family socio-
182 economic status (SES) at the acute time-point. Ratings are made on a seven-point scale
183 where a higher score denotes lower SES.

184

185 *10 years post injury.*

186 Based on previous findings from the longitudinal study two measures of executive
187 functioning were used to explore its role in prediction of problem behaviour [57]. The *Behavior*
188 *Rating Inventory of Executive Function* (BRIEF) [58], Metacognition and Behavioral
189 Regulation index scores, and the General Executive Composite Score (M = 50, SD = 10)
190 were calculated on the basis of parent or close other ratings at the 10 year time-point. Higher
191 scores represent greater dysfunction, and scores >65 indicate functioning at a level of clinical
192 concern.

193 The *20 Questions Task* from the DKEFS test (20Q) [59] was used as a direct assessment
194 of abstract reasoning as it measures abstract thinking as well as problem solving and the
195 utilization of feedback. The abstraction scaled score was used in analyses (M = 10, SD = 3).

196

197 *16 years post-injury.*

198 The *Adult Behavior Checklist* (ABCL) [60] consists of 126 behavior problem items that
199 are evaluated by a significant other for the preceding six months. Statements are scored on a
200 three-level rating scale ranging from not true to very true (M=50; SD=10; borderline/clinical
201 range if score ≥65 for the syndrome scales; ≥60 for the domain) with a higher score
202 indication of greater impairments. As the outcome measure in the present study, the domain
203 Externalizing Behavior was used. It comprises three syndrome scales: Rule Breaking (13
204 items; e.g., gets drunk, in trouble with law), Aggressive Behavior (16 items; e.g., mean to
205 others, threatens people) and Intrusive Behavior (6 items; e.g., brags, demands attention,
206 shows off). The ABCL has been proven reliable in terms of test-retest correlations and
207 internal consistency of scales [60], and has good inter-rater reliability for most scales [61].
208 The *Latrobe Communication Questionnaire* (LCQ) [62] is a 30-item subjective
209 assessment that reflects the four domains Quantity, Quality, Relation and Manner of everyday
210 communication. Each item has four levels of response ranging from (1) "Never or rarely" to
211 (4) "Usually or always" with a higher score reflecting more frequent communication
212 difficulty. Data analyses employed a total score, reflecting overall communication perceived
213 by a significant other.
214 The *Advanced Clinical Solutions Social Perception subtest* (ACS) [63] measures skills
215 associated with the comprehension of social communication. It consists of three emotion
216 perception tests yielding four scores: Affect Naming, Prosody-Faces, Prosody-Pairs, and,
217 collectively, the Emotion Perception Total score. Age-adjusted scaled scores (M=10, SD=3)
218 for each of these test scores were employed in analyses.
219 In addition, respondents were asked whether they had ‘received intervention of any
220 kind (e.g. speech and language, motor, cognitive)’. Those who stated that they had received
221 an intervention were asked to specify the intervention through an open-ended response.
222

223 Procedure

224 The current study was approved by the Human Research Ethics Committee of RCH,
225 Melbourne, Australia. Children were enrolled in the study during their initial hospital
226 admission, and were evaluated at various time points: 0-3 (acute), 6, 12, and 30 months, and
227 5, 10 and 16-years post injury. At each wave of data collection, young people and families
228 enrolled in the original study were sent tracing letters that included a detailed description of
229 the study, and were asked to provide written informed consent, in keeping with hospital
230 ethics procedures. Neuropsychological assessments and questionnaires were administered at
231 each time point by a qualified child psychologist over a two hour period.

232

233 Data analysis

234 All data were entered into SPSS statistical software (Version 21.0; SPSS, Inc.,
235 Chicago, IL) and screened for violations of normality. An alpha level of $p < 0.05$ was used to
236 indicate significance, and effect sizes were calculated using Cohen's *d*.

237 The calculation of individual impairment ratings was based on the ABCL
238 Externalizing Behavior composite scale, and Chi-square tests were employed to examine the
239 proportion young adults in each severity group demonstrating impairment at 16–years post
240 injury. For the broadband scales, scores > 63 are considered clinically significant and scores
241 of 60-63 are in the borderline range for clinical significance.

242 Predictors of 16-year behavioral outcomes were examined using a series of regression
243 analyses as follows;

244 (1). Preliminary univariate regressions were employed to examine relations between
245 EB and all independent variables. Variables that were unrelated to EB at this step were
246 excluded from subsequent analyses.

247 (2). Four separate multivariate adjusted regression models were employed to

investigate relationships between EB and variables related to (a) pre-injury adaptive functioning; (b) injury-related factors/acute intellectual functioning; (c) executive functioning/interventions at 10-years post-TBI; and (d) social cognition and communication at 16-years post injury.

(3). Variables that remained statistically significant in each of the adjusted models were entered into the final adjusted multivariate regression model.

Results

3.1. Demographic and injury characteristics.

There were no significant differences across severity groups with respect to age at 16-year assessment, age-at-injury, pre-injury adaptive abilities, FSIQ, SES or family function (Table 1). Groups differed on gender ($\chi^2(2, 55) = 7.51$, $p = .023$), such that there were a significantly greater proportion of males in the severe TBI group than the mild and moderate TBI groups. As expected, all severity groups differed for GCS-24 hours, $F(2,48) = 21.92$, $p<.001$.

Table 1 about here

3.2. Externalizing symptoms at 16-years post-TBI.

Table 2 displays the total number and proportion of TBI participants found to have clinical or sub-threshold levels of externalizing symptoms. Pearson Chi Square analyses revealed no significant association between externalizing symptoms and injury severity, $\chi^2(2, 55) = .20$, $p = .91$.

Table 2 about here

3.3. Predictors of ABCL Externalizing: Pre-injury adaptive functioning.

Unadjusted model. Preliminary univariate regression analyses revealed significant relationships between ABCL Externalizing and pre-injury Vineland Adaptive Functioning (p=.004), including the Daily Living Skills (p=.040), and Socialization (p=.007). There was no significant relations between ABCL Externalizing and Vineland Communication (p=.106), Family SES (p=.724) or Family Intimacy (p =.188).

Adjusted model. The multivariate adjusted model was moderately significant $F(3,45) = 3.27$, $p = .03$, however due to high colinearity between the independent variables, there were no significant individual pre-injury predictors of ABCL Externalizing (Table 3).

Table 3 about here

3.4. Predictors of ABCL Externalizing: Injury-related factors and acute intellectual functioning.

Univariate regression analysis revealed a significant relation between ABCL Externalizing and FSIQ Time 1, $F(1,42) = 11.80$, p =. 001. (Table 4). There was no significant associations between ABCL Externalizing and Glasgow Coma Score (GCS; p = .491), age at injury (p = .287), neurological signs (p = .363) or surgical intervention (p = .577).

Table 4 about here

3.5. Predictors of ABCL Externalizing: Executive functioning/interventions at 10-years post-TBI.

Unadjusted model. Univariate regression analyses revealed significant relations between ABCL Externalizing and BRIEF Behavioural Regulation Index (p =.026) and 20-questions Abstract Reasoning, (p =.031).

ABCL Externalizing was not significantly associated with BRIEF Global Executive Composite (p=.066), BRIEF Metacognition (p =.128) or access to interventions by 10-years post-injury (p = .427).

Adjusted model. The multivariate adjusted model was moderately significant $F(2,27) = 4.90$, p=.02, with BRIEF Behaviour Regulation Index the single significant predictor (Table 5).

Table 5 about here

3.6. Predictors of ABCL Externalizing: Socio-affective functioning.

Unadjusted model. Univariate regression analyses revealed significant relations between ABCL Externalizing and ACS Social Perception Total Score (p =.02) and LCQ Proxy Report (p <.001).

Adjusted model. The multivariate adjusted model was highly significant $F(2,39) = 10.11$, p<.001, with LCQ Proxy report the single significant predictor (Table 6).

Table 6 about here

3.7. Predictors of ABCL Externalizing: Final adjusted model.

Due to high colinearity between measures of pre-injury adaptive functioning (Table 3), the final adjusted model evaluated the respective contributions of each pre-injury variable via three separate multivariate regression models.

As shown in Table 7, Model 1 was highly significant, $F(4,36) = 9.03$, p<.001, with more frequent externalizing behavior related to poorer adaptive functioning, lower FSIQ (time 1) and more frequent social communication difficulty.

Model 2 ($F(4,36) = 9.03$, $p<.001$) and Model 3 ($F(4,38) = 9.97$, $p<.001$) were highly significant. In addition to the significant predictors identified in Model 1, pre-injury daily living skills and pre-injury socialization emerged as significant individual predictors of externalizing outcome in Model 2 and Model 3 respectively.

Table 7 about here

Discussion

The aim of the present longitudinal prospective study was to 1) investigate rates of EB in young adults with pediatric TBI; and 2) evaluate the respective contributions of a variety of injury and non-injury related risk and resilience factors at various time points to variability in EB outcomes 16 years after pediatric TBI.

There was partial support for all hypotheses. Relative to population expectations, rates of EB were significantly elevated among young adult survivors of pediatric TBI. Moreover, more frequent EB at 16-years post-injury was associated with poorer pre-injury adaptive functioning, reduced acute intellectual functioning and poorer pragmatic communication skills.

Outcomes

Our results show that, by young adulthood, one of every four young people with a history of pediatric TBI had developed clinical or sub-threshold levels of externalising behaviour. Rates of EB in our sample compare to reported prevalence rates of 5-10% in the general population [64,65], indicating a heightened risk of EB in young adulthood following pediatric TBI and mirroring previously identified associations between pediatric TBI and life-course persistent offending behaviors [13].

In keeping with the premises of the HMSC model, EB was linked to a range of pre-injury and post-injury risk factors, discernible in the acute, adolescent and young adult phases. The contribution of pre-injury adaptive functioning to very long term social outcome is consistent with previous reports [66], and may indicate that the influence of early brain injury interacts with a pre-existing vulnerability (i.e., '*double hazard theory*' [67]) to heighten risk for maladaptive behavior in the very-long-term post TBI. Moreover, the relationship between EB and FSIQ converges with previous literature in non-clinical samples [68,69] to suggest that higher levels of intellectual functioning may represent a source of resilience that buffers the risk of behavioral dysfunction in the long-term post injury.

In keeping with previous research [30], pragmatic communication was the strongest and most significant predictor EB in young adults with pediatric TBI. The finding that poorer pragmatic communication was associated with more frequent EB may be interpreted from a diathesis stress perspective [47]. In line with the HMSC model, it may be that difficulty using and ascribing meaning to everyday social discourse contributes to rejection or alienation by interactive partners at the level of the social interaction. In this context, failure to negotiate the complex demands of everyday discourse is likely to elicit distress, reflected in EBs (e.g., aggression, rule breaking, intrusive conduct) which further limit the individual's capacity to negotiate the normative developmental goals of young adulthood.

Contrary to expectations, clinical or sub-threshold levels of EB problems were not associated with injury severity but were instead equally apparent across young adults with TBI of all severity levels. This finding is counter to previous studies that link more severe TBI to elevated risk for violent offending [6,70] and custodial sentences [5,9], and suggests that early clinical indicators of injury severity have limited prognostic utility for longer term behavioral outcomes at least where injuries are sustained during childhood, where the brain is rapidly developing and has potential for reorganization.

Moreover, while previous reports have linked EB and/or persistent offending to factors such as social disadvantage and poorer executive functioning [32-34], these factors did not significantly contribute to EB in the final adjusted model. While these non-significant relationships may to some extent reflect small sample size, they may also indicate that, after prolonged recovery and increasing time since injury, executive function and indices of pre-injury environment become less important prognostic indicators of outcome.

Limitations

Sample. Attrition and sample bias are potential risks with this prospective, longitudinal study. Due to work commitments, travel distance or current life events, some young adult participants were not able to participate. Nevertheless, comparison of the participating and non-participating families indicated no systematic differences with the exception that the non-participant group had lower SES.

Measures. The source of information, proxy-report completed by the significant (either parent or partner/close friend), may also affect the level of reported externalizing symptoms. As young people become more independent, parents may be less knowledgeable about the young person's psychosocial functioning. While Green et al. [71] reported a fair-to-excellent agreement on psychosocial functioning between the adolescent with a pediatric TBI and their parent, Rosema et al. [72] showed that, during the transition into adulthood, the young adult with pediatric TBI did agree with parent report on the more observable behaviors, such as drug and alcohol use, social and communication skills, however, they did not concur on levels of internalizing symptoms, aggressive behavior and thought problems. Therefore self-report as well as direct such as a structured interview is recommended to obtain a more complete representation of risk and resilience factors of EBs following pediatric TBI.

Clinical implications and future research

Relative to population expectations [64,65], a greater proportion of young adults with TBI demonstrated clinical or sub-threshold levels of EBs, which may in turn place these young people at greater risk for maladaptive behavior trajectories characterized by rule breaking, anti-social behaviour and offending. These findings have implications for policy implementation in the youth justice system. For example, screening and assessment for TBI within youth justice services may increase understanding of factors that may lead young people to offend, and assist in identifying young offenders who may benefit from relevant interventions, such as psycho-education and rehabilitation programs that specifically target social communicative dysfunction that persists into the long-term post injury [30]. Surprisingly, and in contrast to the largely medical model used to predict outcomes post-injury, injury characteristics were less important than environmental and pre-injury factors in determining outcome. Environmental factors, in particular, may be seen as potentially modifiable risk factors, offering an opportunity for early intervention to reduce risk of long term problems in this group.

The heightened prevalence of clinical or sub-threshold levels of EB in our sample underscores the need for provision of such early preventative interventions, as well as long term follow up and psycho-education for young people with TBI. For example, initial assessments in the acute and chronic stages of injury may assist to identify children presenting with risk factors, such as poorer pre-injury adaptive functioning and reduced IQ, and direct these children to appropriate services. An awareness of such factors should be shared with primary health care providers and schools, so as to support follow-up provision and further monitoring of relevant factors. This will also offer the means to provide continued engagement with parents and young people regarding the potential medium and

long term impact of TBI on behavioural and functional difficulties that they may not readily associate with the injury.

In addition, further research is needed to identify factors that may be protective against problematic levels of externalising behaviour despite the presence of pre-injury and acute risk factors. Person-oriented, qualitative case study approaches can complement group-level analyses, and offer an opportunity to evaluate how injury and non-injury related risk and resilience factors may interact to contribute to externalising behaviour in young adulthood.

Conclusion

In summary, results suggest that young adults with pediatric TBI are at elevated risk for externalizing trajectories characterized by aggression, rule breaking and intrusive conduct. In line with the HMSC model, more frequent externalizing behaviour was linked to a range of pre-and post injury risk and resilience factors, including poorer pre-injury adaptive functioning, reduced IQ and more frequent pragmatic communication difficulty. These findings underscore the need for screening and assessment of TBI among young offenders, and suggest that early and long-term targeted interventions may be required to address risk factors for EBs in children and young people with TBI.

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Table 1. Characteristics of the TBI sample according to injury severity.

	Mild TBI (n = 15)	Moderate TBI (n = 29)	Severe TBI (n = 11)
Demographics			
No. males, *n* (%)[a]	8 (53)	15 (52)	7 (64)
Injury age (years), *M* (*SD*)	7.89 (3.68)	6.37 (3.38)	6.27 (3.36)
Age at 16-year follow up, (years), *M* (*SD*)	24.49 (4.24)	23.66 (3.63)	23.41 (4.02)
Pre-injury function			
VABS: PRE, *M* (*SD*)	111.64 (20.22)	111.67 (16.15)	106.73 (16.75)
FFQ: PRE, *M* (*SD*)	62.62 (13.99)	65.10 (5.31)	66.55 (5.09)
SES: acute, *M* (*SD*)	3.85 (.93)	4.45 (1.06)	3.97 (1.08)
Acute injury factors			
GCS 24-hours, *M* (*SD*)[a]	14.83 (.39)	12.85 (2.40)	8.90 (2.51)
FSIQ: Acute, *M* (*SD*)	100.93 (13.86)	102.85 (15.33)	97.67 (16.27)

[a] Denotes statistical significance, $p<.05$.

Table 2. Proportion of participants with clinical or sub-threshold levels of externalizing symptoms as a function of injury severity.

	Mild TBI (n = 15)	Moderate TBI (n = 29)	Severe TBI (n = 11)	Total (n = 55)
Impaired, *n* (%)	3 (20)	7 (24)	3 (27)	13 (24)

Table 3. Predictors of ABCL Externalizing: Pre-injury adaptive functioning.

	ABCL Externalizing			
	B	*SE*	*p* value	95% CI
Pre-injury Adaptive function	-.11	.15	.44	[-.41, .18]
Pre-injury Daily Living	.02	.13	.90	[-.24, .27]
Pre-injury Socialization	-.07	.10	.47	[-.28, .13]

Table 4. Predictors of ABCL Externalizing: Acute Intellectual Functioning

	ABCL Externalizing			
	B	*SE*	*p* value	95% CI
FSIQ Time 1	-.22	.06	.001	[-.35, -.09]

Table 5. Predictors of ABCL Externalizing: Executive function at 10-years post-TBI.

	ABCL Externalizing			
	B	*SE*	*p* value	95% CI
BRIEF BR	.16	.08	.05	[.01, .33]
20-questions	-.48	.31	.13	[-1.11, .16]

Table 6. Predictors of ABCL Externalizing: Socio-affective function at 16-years post-TBI.

	ABCL Externalizing			
	B	SE	*p* value	95% CI
ACS Social Perception	-.29	.21	.18	[-.72, .14]
LCQ Proxy Report	.35	.10	.01	[.14, .56]

Table 7: Predictors of ABCL Externalizing: Final Adjusted Model.

	ABCL Externalizing			
	B	*SE*	*p* value	95% CI
Model 1				
Pre-injury Adaptive Composite	-.09	.05	.05*	[-.19, .00]
FSIQ Acute	-.13	.06	.02*	[-.25, -.02]
BRIEF BR 10-years	.08	.07	.26	[-.06. .22]
LCQ Proxy 15-years	.28	.09	.003*	[.11, .46]
Model 2				
Pre-injury Daily Living Skills	-.11	.05	.05*	[-.23, -.01]
FSIQ Acute	-.15	.05	.01*	[-.26 -.04]
BRIEF BR 10-years	.09	.07	.22	[-.06, .24]
LCQ Proxy 15-years	.28	.09	.003*	[.10, .46]
Model 3				
Pre-injury Socialization	-.10	.04	.04*	[-.19, -.01]
FSIQ Acute	-.14	.06	.02*	[-.25, -.02]
BRIEF BR 10-years	.08	.06	.25	[-.06, .21]
LCQ Proxy 15-years	.29	.08	.002*	[.12, .46]

*Denotes statistically significant relationship, $p<.05$.